Dissertation on

A COMPARATIVE EVALUATION OF PROPOFOL AND SEVOFLURANE BASED ANAESTHETIC TECHNIQUE ON PERI-OPERATIVE PARAMETERS OF PATIENTS UNDERGOING FESS UNDER HYPOTENSIVE ANAESTHESIA

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Certificate

This is to certify that the dissertation entitled, "A comparative evaluation of propofol and sevoflurane based anaesthetic technique on peri-operative parameters of patients undergoing fess under Hypotensive Anaesthesia" submitted by Dr.S.Merlin Shalini Ruth, in partial fulfilment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr. M.G.R. Medical University, Chennai is a bonafide record of the work done by her in the Department of Anaesthesiology, Madras Medical College, during the year 2006 – 2009.

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CONTENTS

CHAPTER I

Introduction

Anaesthesia for Functional endoscopic sinus surgery is a challenging job. The surgeons' operating field itself is very small and surrounded with mucus membranes. It is imperative for the surgeons to look at a clear surgical field in order to identify the diseased tissue properly. A small amount of blood within the field is enough to occlude the view through the endoscope making things difficult for the surgeon and incomplete removal of the diseased tissue will cause the disease to reoccur. Anaesthesiologists have devised various techniques to prevent this bleeding, of which induced hypotension has stood the test of time. This surgery per se is not a major one by its standards and surgeons recently have been trying to accomplish FESS as a day care surgery. Different anaesthetic techniques using different pharmacological agents have been used to induce controlled hypotension. I have chosen to study and compare the effects of inhalational agent – sevoflurane based anaesthesia and intravenous agent – propofol based anaesthesia in reducing the intra operative blood loss.

This study proposes to analyse the anaesthetic challenges of keeping the operating field free of blood through pharmacological therapy. It serves to study whether the traditional inhalational agent based anaesthesia – now sevoflurane anaesthesia in

our department scores over propofol based anaesthesia in providing better endoscopic vision for the surgeon.

CHAPTER II

AIM

To evaluate the effect of propofol versus sevoflurane anaesthesia as maintenance anaesthetics on the perioperative parameters of patients undergoing FESS under GA and controlled hypotension technique.

The following parameters are compared:

1. Ability to achieve targeted MAP
2. Hemodynamic stability
3. Undesirable side effects
4. Blood loss
5. Endoscopic vision

CHAPTER III

Hypotensive anaesthesia

Hypotensive anaesthesia is a technique, used intra operatively to help minimize surgical blood loss, thereby decreasing the need for blood transfusion and also to provide a clear surgical field. The technique entails the controlled lowering of blood pressure and is defined as a reduction of the systolic blood pressure to between 80-90 mmHg. An alternative definition is a decrease in the mean arterial pressure (MAP) to 50-70 mmHg in a normotensive patient.

Inducing hypotension

Deliberate hypotension is induced by a variety of pharmacological agents and non pharmacological methods. Since there is no single agent capable of safely and effectively lowering arterial pressure in all situations, the anaesthetist may need to employ a variety of agents or techniques in order to achieve the target pressure.

Pharmacological agents can generally be divided into two categories: peripheral vasodilators and inhalation agents.

The three most commonly used vasodilators are: sodium nitroprusside (SNP), nitroglycerine (NTG) and trimethaphan.

SNP acts as a vascular smooth muscle relaxant and has a rapid onset but brief duration of action. Its primary influence is on arteriolar and venous vessels, but without significant myocardial effects.

NTG reduces blood pressure by relaxing venous smooth muscle and, like SNP, has rapid onset of action but short duration. NTG is less toxic than SNP. However, it is less potent than SNP in its capacity to reduce blood pressure.

Trimethaphan produces hypotension through ganglionic blockade and direct vasodilator properties. It is also short acting and provides tight control of blood pressure.

Commonly used inhalation agents, or volatile anaesthetic agents, include halothane, isoflurane and sevoflurane. The concentration of a volatile anaesthetic agent produces a dose dependent decrease in mean arterial pressure.

Alpha2 agonists and beta blockers are also used for this purpose. Spinal and epidural anaesthesia can also be used to produce controlled hypotension. Unfortunately, these techniques require large infusions of fluids and the deliberate hypotension can be erratic and difficult to control.

Indications to hypotensive anaesthesia

- Excision of intracranial tumors
- Aneurysm excisions (Cerebral, carotid, aortic)
- FESS procedure
- Middle ear surgeries
- Spine/hip surgeries

Contraindications to hypotensive anaesthesia

- Congenital heart disease
- Severe anaemia
- Coronary artery disease

- Congestive cardiac failure
- Poorly controlled hypertension
- Increased intracranial pressure
- Significant cerebro-vascular disease
- Low flow states to the liver or kidney

CHAPTER IV

Functional Endoscopic Sinus Surgery

Rhinology and sinus surgery have undergone a tremendous expansion since the discourses of Messerklinger and Wigand in the late 1970s. Imaging advances, increased understanding of the anatomy and the pathophysiology of chronic sinusitis, and image-guided surgery have allowed surgeons to perform more complex procedures with increased safety.

Functional endoscopic sinus surgery is the primary approach used today for the surgical treatment of chronic sinusitis.

Indications

Endoscopic sinus surgery is most commonly performed for inflammatory and infectious sinus disease. The most common indications for endoscopic sinus surgery are as follows:

- Chronic sinusitis refractory to medical treatment
- Recurrent sinusitis

- Nasal polyposis
- Antrochoanal polyps
- Sinus mucocoeles
- Excision of selected tumours
- Cerebrospinal fluid (CSF) leak closure
- Orbital decompression (e.g., Grave’s ophthalmopathy)
- Optic nerve decompression
- Dacryocystorhinostomy (DCR)
- Choanal atresia repair
- Foreign body removal
- Epistaxis control

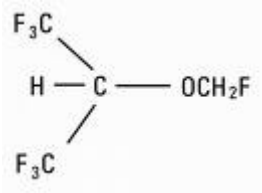
FESS is a delicate and time consuming procedure. It is performed routinely under general anaesthesia. Anaesthesiologists have to plan the technique in such a way that will facilitate the operating team for achieving a bloodless field for better visualization of the intranasal structures and minimize intra operative bleeding,

because even minimal bleeding can obstruct the view of the operating endoscope. Hence comes the role of hypotensive anaesthesia. Many pharmacological agents, including sevoflurane as a combination are in use for inducing intra operative hypotension. It is easy to administer and control its concentration, does not require infusion pump and can be quickly washed out from body by hyperventilation. As sevoflurane induces hypotension slower than the other pharmacological agents like sodium nitroprusside, continuous intra-arterial blood pressure monitoring is not mandatory. It causes least arrhythmia in presence of exogenous adrenaline which is routinely infiltrated into the operative field for creating a bloodless field.

CHAPTER V

PHARMACOLOGY OF THE STUDY DRUGS

SEVOFLURANE



Sevoflurane is fluorinated methyl isopropyl ether used for inhalational anaesthesia.

Physical properties

Molecular weight - 200 g/mol

Boiling point at 1 atm - 58.5º C

Vapour pressure at 20ºC - 170 mmHg

Partition coefficient at 37ºC

Blood – Gas - 0.69

Brain – Blood - 1.7

Fat – Blood - 48

Oil – Gas - 47.2

MAC at P_B 760 mmHg.

30-55 years of age - 1.8%

For children

0-1 month - 3.3%

1-6 months - 3.0%

6 months – 3 years - 2.8%

3-12 years - 2.5%

Mechanism of action of inhaled anaesthetics

Inhaled anaesthetics act in different ways at the level of the central nervous system. They may disrupt normal synaptic transmission by interfering with the release of neurotransmitters from presynaptic nerve terminal (enhance or depress inhibitory or excitatory transmission), by altering the re-uptake of neurotransmitters to the post synaptic receptor sites or by influencing the ionic conductance change that follows activation of the post synaptic receptor by neurotransmitters. Both pre and post synaptic effects have been found.

Direct interaction with the neuronal plasma membrane is very likely, but indirect action via production of a second messenger also remains possible. The high correlation between lipid solubility and anaesthetic potency suggests that inhalation anaesthetics have a hydrophobic site of action. Inhalation agents may bind to both membrane lipids and proteins. It is at this time not clear which of the different theories are most likely to be the main mechanism of action of inhalation anaesthetics.

The Meyer - Overton theory describes the correlation between lipid solubility of inhaled anaesthetics and MAC suggests that anaesthesia occurs when a significant number of inhalation anaesthetic molecules dissolve in the lipid cell membrane. The Meyer – Overton rule postulates that the number of molecules dissolved in the lipid cell membrane and not the type of inhalation agent causes anaesthesia. Combinations of different inhaled anaesthetics may have additive effects at the level of the cell membrane.

However, the Meyer – Overton theory does not describe why anaesthesia occurs. Mullins expanded the Meyer – Overton rule by adding the so-called Critical Volume Hypothesis. He states that the absorption of anaesthetic molecules could

expand the volume of a hydrophobic region within the cell membrane and subsequently distort channels necessary for sodium ion flux and the development of action potentials necessary for synaptic transmission. The fact that anaesthesia occurs with significant increase in volume of hydrophobic solvents and is reversible by compressing the volume of the expanded hydrophobic region of the cell membrane supports Mullins critical volume hypothesis.

The protein receptor hypothesis postulates that protein receptors in the central nervous system are responsible for the mechanism of action of inhaled anaesthetics. This theory is supported by the steep dose response curve for inhaled anaesthetics. However, it remains unclear if inhaled agents disrupt ion flow through membrane channels by an indirect action on the lipid membrane via a second messenger or by direct and specific binding to channel proteins.

Another theory describes the activation of Gama Amino Butyric Acid (GABA) receptors by the inhalation anaesthetics. Volatile agents may activate GABA channels and hyperpolarize cell membranes. In addition, they may inhibit certain calcium channels and therefore prevent release of neurotransmitters and inhibit glutamate channels. Volatile anaesthetics share therefore common cellular actions with other sedative, hypnotic or analgesic drugs.

The true mechanism of action of volatile anaesthetics may be a combination of two or more such theories described as multisite action hypothesis.

Pharmacokinetics

Uptake and distribution of inhaled anaesthetics:

A series of partial pressure gradients, beginning at the vaporizer of the anaesthetic machine, continuing in the anaesthetic breathing circuit, the alveolar tree, blood and tissue will ensure the forward movement of the gas. The principal objectives of that movement are to achieve equal partial pressure of the gas. After a short period of equilibration the alveolar partial pressure equals the brain partial pressure. Alveolar partial pressure can be raised by increasing minute ventilation, flow rates at the level of the vapourizer and by using a non-rebreathing circuit.

Two special effects increasing the amount of gas in the alveoli have to be mentioned. The concentration effect describes how the concentration of the gas in the remaining alveolar volume can increase after some of the gas has been transferred into the blood. The second gas effect usually refers to nitrous oxide combined with an inhalation agent. Because nitrous oxide is not soluble in blood, its rapid absorption from alveoli causes an abrupt rise in the alveolar concentration

of the other inhalation anaesthetic. All the above mentioned factors influence the inflow of gas into the alveoli.

Solubility, cardiac output and the alveolar to venous anaesthetic gradient represent outflow factors. Inflow factors minus outflow factors equals alveolar partial pressure of the gas.

Solubility describes the affinity of the gas for a medium such as blood or fat tissue.

The blood : gas partition coefficient describes how the gas will partition itself between the two phases after equilibrium has been reached. The blood : gas coefficient of sevoflurane (0.69) ensures prompt induction of anaesthesia after discontinuation of the anaesthetic. Compared with isoflurane, recovery from sevoflurane anaesthesia is faster and the difference is magnified in longer duration surgical procedures (>3 hours).

A higher cardiac output removes more volatile anaesthetic from the alveoli and lowers therefore the alveolar partial pressure of the gas. The agent might be faster distributed within the body but the partial pressure in the arterial blood is lower. It

will take longer for the gas to reach equilibrium between the alveoli and the brain. Therefore, a high cardiac output prolongs induction time.

The alveolar to venous partial pressure difference reflects tissue uptake of the inhaled anaesthetics. A large difference is caused by increased uptake of the gas during the induction phase. This facilitates the diffusion of the gas from the alveoli into the blood. The brain : blood coefficient describes how the gas will partition itself between the two phases after equilibrium has been reached. Sevoflurane for example has a brain : blood coefficient of 1.7 meaning that if the gas is in equilibrium, the concentration in the brain will be 0.7 times higher than the concentration in the blood. All inhalation anaesthetics have high fat : blood partition coefficients. This enormous capacity of fat for anaesthetic means that most of the anaesthetic contained in the blood perfusing fat is transferred to the fat. Although most of the anaesthetic moves from the blood into the fat, the anaesthetic partial pressure in fat increases very slowly. The large capacity of fat and its low perfusion per milliliter explains the delayed recovery in obese patients.

Pharmacodynamics

Central nervous system

Inhaled anaesthetics cause loss of response to verbal commands at MAC-awake concentrations. Surgical stimulation increases the anaesthetic requirement to prevent awareness.

Volatile anaesthetics produce dose dependent increases in cerebral blood flow (CBF). Sevoflurane has an intrinsic dose dependent cerebral vasodilatory effect. Sevoflurane does not alter auto regulation of CBF.

Inhaled anaesthetics produce increases in intracranial pressure that parallel increases in CBF produced by them.

Inhaled anaesthetics produce dose dependent decreases in cerebral metabolic oxygen requirements (CMR O_2). When EEG becomes isoelectric, an additional increase in the concentration of the volatile anaesthetics does not produce further decreases in CMR O_2.

Cardiovascular system

Volatile anaesthetics produce dose dependent decreases in mean arterial pressure. The decrease produced by sevoflurane principally results from a decrease in systemic vascular resistance.

Sevoflurane increases heart rate only at concentrations of >1.5 MAC. A small dose of opioid (morphine in the preoperative medication or fentanyl intravenously immediately before induction of anaesthesia) can prevent the heart rate increase associated with volatile anaesthetics.

Volatile anaesthetics exert little or no predictable effect on pulmonary vascular resistance.

Sevoflurane has no effect on the atrioventricular or accessory pathways and is considered an acceptable anaesthetic drug for patients undergoing ablative procedures.

Volatile anaesthetics induce coronary vasodilatation by preferential action on vessels with diameters from 20µ to 50µ. They are cardio protective.

Respiratory system

Volatile anaesthetics produce dose dependent increases in the frequency of breathing and decreases in tidal volume. The net effect is a rapid and shallow pattern of breathing. The increase in frequency of breathing is insufficient to offset decreases in tidal volume, leading to decreases in minute ventilation and increases in $PaCO_2$.

Sevoflurane decreases the ventilator response to carbon di oxide. It produces apnea between 1-5 and 2-0 MAC. All inhaled anaesthetics profoundly depress the ventilator response to hypoxemia.

Sevoflurane produces bronchodilatation in normal individuals and in patients with COPD.

Skeletal muscles

Sevoflurane and other fluorinated ethers produce skeletal muscle relaxation and produce dose-dependent enhancement of the effects of neuromuscular blocking drugs.

Obstetrics

Volatile anaesthetics produce dose dependent decreases in uterine smooth muscle contractility. This is desirable to facilitate removal of retained placenta. Conversely, uterine relaxation produced by volatile anaesthetics may contribute to blood loss due to uterine atony.

Metabolism

3 to 5% of the dose administered undergoes oxidative metabolism by cytochrome P-450 enzymes to form organic and inorganic fluoride metabolites. In addition, sevoflurane is degraded by dessicated carbon dioxide absorbents containing strong bases to potentially toxic compounds especially when the temperature is increased. Among these compounds, only compound A – Triflurомethyl vinyl ether (and to a lesser extent compound B) are encountered clinically. Compound A is a dose dependent nephrotoxin in animals. Although this finding is a concern, the levels of these compounds (particularly compound A) that occur during administration of sevoflurane to patients are far below speculated toxic levels.

The rationale for utilizing at least 2litre/minute of fresh gas flow rate when administering sevoflurane is intended to minimize the concentration of compound

A that may accumulate in the anaesthesia breathing circuit to assess the adequacy of this recommendation, the concentration of compound A of anaesthesia with 1.25 MAC sevoflurane during 2 to 8 hours was found to be in the range of 40 to 42 ppm which is far below the toxic levels.

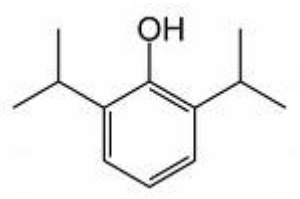
In children, sevoflurane anaesthesia lasting for 4 hours using total fresh gas flows of 2 litre/minute produced concentrations of compound A of <15 ppm and there was no evidence of renal dysfunction.

A proposed mechanism of nephrotoxicity is metabolism of compound A via beta-lyase pathway to a reactive thiol. Because humans have less than one-tenth of the enzymatic activity for this pathway compared to rats, it is possible that humans should be less vulnerable to injury by this mechanism.

Probenecid is a selective inhibitor of organic anion transport and pretreatment with this drug prevents compound A induced renal injury in animals and may provide similar protection in humans.

Sevoflurane is non pungent, has minimal odour and causes the least degree of airway irritation among the currently available volatile anaesthetics. For these reasons, sevoflurane is acceptable for inhalation induction of anaesthesia.

PROPOFOL



Propofol is a substituted isopropyl phenol (2,6 – di isopropyl phenol) that is administered intravenously as 1% solution in an aqueous solution of 10% soybean oil, 2.25% glycerol and 1.2% purified egg phosphatide. This drug is chemically distinct from all other drugs that act as intravenous sedative – hypnotics.

Administration of propofol, 1.5 to 2.5 mg/kg as a rapid IV injection (<15 sec), produces unconsciousness within about 30 seconds. Awakening is more rapid and complete than that after induction of anaesthesia with all other drugs.

The more rapid return of consciousness with minimal residual central nervous system effects is one of the most important advantages of propofol.

Mechanism of action

Propofol is presumed to exert it's sedative – hypnotic effects through an interaction with GABA receptor, the principal inhibitory neurotransmitter in the central nervous system. When GABA receptor is activated, transmembrane chloride conductance increases, resulting in hyprepolarisation of the post synaptic cell membrane and functional inhibition of the post synaptic neuron. The interaction of propofol with specific components of $GABA_A$ receptors appears to decrease the rate of dissociation of the inhibitory neurotransmitter, GABA from the receptor, thereby increasing the duration of the GABA activated opening of the chloride channel with resulting hyper polarization of cell membranes.

Pharmacokinetics

Propofol is rapidly metabolized in the liver by conjugation to glucuronide and sulphate to produce water-soluble compounds which are excreted by the kidneys. Less than 1% of Propofol is excreted unchanged in urine and 2% is excreted in faeces. As clearance of Propofol exceeds hepatic blood flow, extrahepatic metabolism and extrarenal elimination has been suggested. Lungs are responsible for approximately30% of the uptake and first-pass elimination after a bolus dose. During a continuous infusion of Propofol, there is a 20-30% decrease in Propofol

and a higher concentration of the metabolite 2,6-diisopropyl-1,4 quinol on the arterial side of the circulation. Despite the rapid clearance of Propofol by metabolism, there is no evidence of impaired elimination in patients with cirrhosis of the liver. Renal dysfunction does not influence the clearance of Propofol. The rapid clearance of Propofol confirms this drug can be administered as a continuous infusion without an excessive cumulative effect.

By a three-compartment model, initial and slow distribution half-lines are 1 to 8 minutes and 30 to 70 minutes and an elimination half-life of 4 – 23.5 hours. Volume of distribution of central compartment is 20-40 litres and volume of distribution at steady state (Vd_{ss}) is 150 – 700 litres (2-10 lit/kg). Clearance is 20-30 ml/kg/min – 1.5 to 2.2 lit/min. It has a short effect site equilibration time equilibrium constant (K_{ev}) for Propofol based on suppression of EEG is about 0.3 min^{-1}

Pharmacodynamics

Cardiovascular system

Propofol produces decrease in arterial blood pressure during induction of anaesthesia. Induction dose of 2-2.5 mg/kg produces a 25-40% reduction in blood

pressure. Similar changes are seen in diastolic and mean blood pressure. The decrease in arterial pressure is associated with a decrease in cardiac output by 15% and decrease in systemic vascular resistance by 15-25%. The reduction in systemic pressure after an induction dose is due to vasodilatation and possibly myocardial depression. At high doses, 10 µg/ml, propofol abolishes the ionotropic effects of alpha but not beta stimulation and enhances lusitropic effect of beta stimulation. Vasodilatory effect is due to reduction in sympathetic activity.

During maintenance of anaesthesia with propofol infusion, systolic pressure remains between 20 and 30% below preinduction levels. In patients receiving narcotic premedication and nitrous oxide with an infusion of propofol 54-104 µg/kg/min, systemic vascular pressure is not significantly decreased from baseline but cardiac output and stroke volume are decreased. Because vasodilatory and myocardial depressant effects are concentration dependant, the decrease in blood pressure from propofol infusion is much less than that seen after an induction bolus. Heart rate may decrease, increase or remain unchanged when anaesthesia is maintained with propofol. Infusion of propofol results in significant reduction in both myocardial blood flow and myocardial oxygen consumption, a finding that suggests preservation of the global myocardial oxygen supply-demand ratio.

Respiratory system

Propofol produces dose dependant depression of ventilation, with apnoea occurring in 25-35% of patients after induction of anaesthesia with propofol. Opioids given as premedication enhance ventilatory depressant effect. A maintenance infusion of propofol decreases tidal volume and respiratory rate.

The ventilatory response to carbon di oxide and arterial hypoxemia are decreased by propofol. The decreased ventilatory response to hypercapnia is due to the effect at the central chemoreceptors. In contrast to low dose volatile anaesthetics, the peripheral chemoreflex response to carbon di oxide remains intact.

Propofol can produce bronchodilatation and decrease the incidence of intra operative wheezing in patients with asthma. Propofol does not cause laryngospasm.

Central nervous system

Propofol is primarily a hypnotic. Induction dose is 1.5 to 2.5 mg/kg. Onset of hypnosis after doses of 2.5 mg/kg is rapid (one arm-brain circulation) 30-40 seconds. Peak effect is seen at 90 to 100 seconds. Duration of hypnosis 5 to 10

min. Propofol decreases cerebral blood flow, cerebral metabolic oxygen consumption and intra cranial pressure. It also increases cerebrovascular resistance but does not appear to affect cerebrovascular reactivity to changes in arterial carbon di oxide tension.

Other systems

Propofol anaesthesia is associated with significant decreases in intraocular pressure by as much as 30-50%. This decrease may be associated with a concomitant decrease in systemic vascular resistance.

Although propofol has the potential for affecting adrenal steroidogenesis, it does not appear to block cortisol and aldosterone secretion in response to surgical stress or adrenocorticotropic hormone in clinical practice. Although transient decreases in plasma cortisol concentration have occurred, these reductions have not been sustained.

Side effects

Allergic reactions

Allergic components of propofol include the phenyl nucleus and di isopropyl side chain. Allergic reactions are described in patients with history of other drug allergies often to neuromuscular blocking drugs.

Lactic acidosis

Also known as “propofol infusion syndrome” is described in patients receiving prolonged high dose infusions of propofol (>75 μ/kg/min) for longer than 24 hours. Unexplained tachycardia during propofol anaesthesia should prompt evaluation.

Other side effects include

1. Abuse potential
2. Bacterial growth – the contents of an opened vial must be discarded if they are not used within 6 hours.

3. Pain on injection – 1 ml 1% lignocaine injection prior to propofol administration decreases the incidence.

Clinical uses

Induction of anaesthesia

The induction dose in healthy adults I s1.5 to 2.5 mg/kg IV, with blood levels of 2 to 6 µg/ml producing unconsciousness depending on associated medications and the patient's age. Awakening typically occurs at plasma propofol concentrations of 1.0 to 1.5 µg/ml.

K_{eo} is the rate constant for equilibration between plasma and the site of drug effect (or) for transfer of drug from the site of drug effect to the environment.

$T_{1/2}$ k_{eo} for propofol is 2.4 minutes.

By knowing the k_{eo} of propofol, we can design a dosing regimen that yields the desired concentration at the site of drug effect.

Maintenance of anaesthesia

Several infusion schemes have been used to achieve adequate plasma concentration of propofol after an induction dose, an infusion of 100-200 µ/kg/min is usually needed. The infusion rate is then titrated to individual requirements and the surgical stimulus. When combined with propofol, fentanyl reduces its required indusion rate and concentration.

Drug conc.	Skin incision	Minor surgery	Major surgery	Awakening	Analgesia or sedation
µg/ml	2-6	2.5-7.5	2-6	0.8-1.8	1-3

In our study, we have followed the Bristol regimen for maintenance of anaesthesia. We have used propofol infusion of 10 mg/kg/hour for the first 10 minutes followed by 8 mg/kg/hour for the next 10 minutes followed by 6 mg/kg/hour for rest of the procedure.

Antiemetic

Sub hypnotic doses of propofol are effective against chemotherapy induced nausea and vomiting. When administered to induce and maintain anaesthesia, it is more effective than ondansetron in preventing PONV. Mechanism is unknown.

Antipruritic

Propofol, 10 mg IV, is effective in the treatment of pruritus associated with neuraxial opioids or cholestasis. The quality of analgesia is not affected by propofol.

CHAPTER VI

REVIEW OF LITERATURE

This study was constructed to evaluate whether propofol or sevoflurane as maintenance of anaesthesia offers any advantage for the conduct of hypotensive anaesthesia technique for patients undergoing FESS procedure under GA. The null hypothesis postulated for testing was that, there would be no difference between propofol and sevoflurane as maintenance anaesthesia in their effects on improved hemodynamic stability to reduce blood loss or improve endoscopic vision. Literature was reviewed to analyse the existence of similar studies.

Sevaci et al[1], in their study on 'Comparison of propofol and sevoflurane anaesthesia by means of blood loss during endoscopic sinus surgery', recruited 32 ASA I or II adult patients. Induction was done in both groups with 2.5 mg/kg propofol, 0.5mg/kg rocuronium bromide and 2 µg/kg fentanyl.In Group 1 , Maintenance was 40/60% O2-air mixture with propofol infusion 12mg/kg/hr, 9mg/kg/hr, 6mg/kg/hr for successive 30 minutes of surgery. They found that blood loss was significantly less in the propofol group**.** In Group 2 , maintenance was with 33/66% O_2-N_2O mixture and 2-2.5% sevoflurane.

Manola et al[2], in their study on ‘Using remifentanil and sufentanil in functional endoscopic sinus surgery to improve surgical conditions’ recruited 71 patients . For maintenance of anesthesia, group A used sufentanil and sevoflurane, group B used remifentanil/propofol infusion and group C used fentanyl and isoflurane. They found that group B scored over group A and C in visibility of the surgical field and quantity of blood loss.

Ahn et al[3], in their study on ‘Comparison of surgical condition during propofol or sevoflurane anaesthesia for endoscopic sinus surgery’ recruited 40 ASA I/II patients. As maintenance, group 1 received propofol/remifentanil infusion and group 2 received sevoflurane/remifentanil infusion. They found that patients in propofol/remifentanil group had lesser blood loss and sevoflurane/remifentanil group.

Wormald et al[4], in their study on ‘The effect of total intravenous anaesthesia compared with inhalational anaesthesia on the surgical field during endoscopic sinus surgery’ recruited 56 ASA I, II patients. As maintenance of GA, group I received sevoflurane with fentanyl, group II received propofol and remifentanil infusion. They found that TIVA results in better surgical field than inhalational anaesthesia.

Tirelli et al[5], in their study 'Total intravenous anaesthesia in endoscopic sinus-nasal surgery' recruited 64 ASA I, II patients. Group I received TIVA using remifentanil and propofol. Group II used sevoflurane and fentanyl. They found that though the hypotensive effects of both groups is equivalent, only total intravenous anaesthesia is effective in reducing bleeding during functional endoscopic sinus-nasal surgery.

Blackwell et al[6], in their study 'Propofol for maintenance of general anaesthesia: a technique to limit blood loss during endoscopic sinus surgery' retrospectively reviewed 25 patients. Group 1 had received propofol infusion for maintenance and group 2 received isoflurane as maintenance anaesthesia. They found that the average estimated blood loss was decreased in group 1.

Eberhart et al[7], in their study 'Intravenous anaesthesia provides optimal surgical conditions during microscopic and endoscopic sinus surgery' recruited 90 patients. Group 1 used propofol and remifentanil 10-30 µg/kg/hr. Group 2 received isoflurane 0.4-1% and repetitive doses of 0.5 to 1mg alfentanyl. An injectable vasodilator was used in both groups to keep mean arterial pressure between 60 and 70mmHg. They found that intravenous anaesthesia using propofol – remifentanil provides better surgical condition compared with isoflurane – alfentanil.

Pavlin et al[8], in their study ‘Propofol versus isoflurane for endoscopic sinus surgery’ recruited 56 patients.Group 1 received propofol, Group 2 received isoflurane, both groups supplemented with nitrous oxide\oxygen and alfentanil. They found that surgical blood loss was same for both anaesthetic agents , but propofol appeared to offer an advantage in terms of subjective improvement in operating conditions.

Beule et al[9], in their study ‘Propofol Vs Sevoflurane: bleeding in endoscopic sinus surgery’, recruited 46 patients. Group1 received maintenance with sevoflurane/fentanyl. Group 2 received maintenance with propofol/fentanyl. They found that under conditions of balanced circulatory parameters, equal blood loss and endoscopic vision can be achieved with both regimens.

Cafiero et al[10], in their study ‘Clinical comparison of remifentanil-sevoflurane Vs remifentanyl-propofol for endoscopic transsphenoidal surgery’, recruited 44 patients. Group P received propofol and remifentanil. Group S received remifentanil and sevoflurane. They found that sevoflurane-remifentanil gives faster recovery and equivalent intra op status when compared with propofol group.

Shigeki et al[11], in their study "comparative evaluation of total intravenous anaesthesia with propofol-fentanyl and thiopental –sevoflurane anaesthesia using laryngeal mask airway for diagnostic bronchoscopy" recruited 60 patients. Group 1 was induced with propofol and fentanyl and maintained with continuous infusion of propofol with fentanyl. Group 2 was induced with thiopentone and maintained with N_2O/O_2/sevoflurane.Insertion of LMA was facilitated with vecuronium and ventilation was controlled. During maintenance of anaesthesia intra operative MAP was found to be lower and was maintained at that value.

Steinmetz et al[12], in their study "Hemodynamic differences between propofol-remifentanil and sevoflurane anaesthesia for repair of cleft lip and palate in infants" recruited 39 infants. Group 1 received a combination of remifentanil and propofol. Group 2 received sevoflurane-fentanyl anaesthesia for surgical repair of cleft lip and palate. They found that remifentanil-propofol infusion was associated with lower heart rates than sevoflurane group.

Zeliha et al[13], in their study 'Comparison of remifentanil-propofol and sevoflurane for preventing cardiovascular response and quality of recovery in pediatric surgery' recruited 30 pediatric patients undergoing elective ENT surgery. They

found that TIVA provided lower preoperative heart rates and blood pressure than sevoflurane based anaesthesia.

Jellish et al[14], in their study 'The comparative effect of sevoflurane versus propofol in the induction and maintenance of anaesthesia in adult patients' recruited 186 ASA I, II patients undergoing elective surgical procedures of 1-3 hours. Group I (n = 93) received sevoflurane-nitrous oxide for both induction and maintenance of anaesthesia while group 2 (n = 93) received propofol-nitrous oxide anaesthesia. They compared induction times, emergence time, complications, side effects and hemodynamic stability. They found that both groups were hemodynamically stable throughout the study period.

Watson et al[15], in their study "Clinical comparison of 'single agent' anaesthesia with sevoflurane versus target controlled infusion of propofol" recruited 40 patients undergoing spinal surgery. Group 1 received propofol-air-oxygen for induction followed by propofol-air-oxygen for maintenance. Group 2 received 8% sevoflurane-oxygen for induction and sevoflurane-oxygen-nitrous oxide for maintenance. They found that cardiovascular stability was good and comparable in both groups.

CHAPTER VII

MATERIALS AND METHODS

After getting clearance from the ethics committee, the study was formulated as follows.

Study design

A prospective, randomized study

Case definition

Inclusion criteria

- ASA I and II
- Patients between ages 16 and 60
- Patients undergoing endoscopic sinus surgery
- Chronic sinusitis
- Sinonasal polyposis

Exclusion criteria

- Hypertensive patients
- IHD
- History of CVA or TIA
- Poor respiratory reserve
- Significant hepatic/renal disease
- Patient refusal
- Hypersensitivity to study drug
- Not satisfying inclusion criteria

Materials and methods

- A prospective, randomized study
- Ethical committee approval
- Informed consent
- 40 patients posted for endoscopic sinus surgery were drafted if they fulfilled the inclusion criteria
- Preoperative evaluation included detailed elicitation of significant history, clinical evaluation

- Preoperative investigation included Hb, PCV, BT, CT, RFT, ECG and chest X-ray.
- Premedication included oral alprazolam 0.5mg 3 hours before surgery
- Any relevant specialist opinion, investigation and care were obtained
- Nasal packing done with 4% lignocaine with oxymetazoline
- In the operation theatre, IV access was obtained with 18G IV cannula. Preloading was done with 10ml/kg of balanced salt solution.
- Monitoring was done using a L&T monitor which included ECG, NIBP, SpO2 and temperature.
- After recording baseline values and preloading, patients were given the drug based on the group they were assigned to.
- Preoxygenation with 100% oxygen for 3 min.
- Induction : Fentanyl 2mcg/kg + Propofol 2.5 mg/kg + Rocuronium 0.6 mg/kg
- Endotracheal intubation
- Throat packing
- In group S, maintenance with 66% N2O, 33% O2, 3% sevoflurane with IPPV and rocuronium as relaxant.

- In group P, maintenance with 66% N2O, 33% O2, propofol 10mg/kg for initial 10 min, 8mg/kg for next 10 min, 6mg/kg till the end of procedure with IPPV and rocuronium as relaxant.

Plan

Plan is to maintain MAP between 60 – 70 mmHg.

If MAP > 70mmHg:

Start a titrated NTG infusion at 0.3µg/kg/min. Increase by 0.3µg/kg/min with an interval of 5 min to allow equilibration of serum therapeutic levels.

If MAP < 60mmHg:

Step 1: IV fluids RL/NS – 200ml

Step 2: Taper down NTG

Step 3: IV Ephedrine 6mg bolus

If HR > 150 bpm:

IV Esmolol bolus 500mcg/kg

If HR < 50 bpm:

IV Atropine 0.6 mg

In case of arrhythmias:

If hemodynamically stable, continue with the study with close and increased monitoring.

If hemodynamically unstable, abandon hypotensive anaesthesia and manage accordingly.

End of procedure:

Reversal with neostigmine 50 µg/kg + glycopyrollate 8 µg/kg and extubation.

Parameters studied

1. Heart rate
2. Systolic blood pressure
3. Diastolic blood pressure
4. Mean arterial pressure
5. Requirement of nitroglycerine
6. Intra operative problems (hypotension, hypertension, arrhythmias, tachycardia, bradycardia and ischemia)
7. Duration of surgery
8. Operating field
9. Intra operative blood loss
10. Post anaesthesia discharge criteria

Post anaesthesia discharge scoring system

Score	0	1	2
Vital signs	>40% of preoperative baseline	20-40% preoperative baseline	Within 20% of preoperative baseline
Activity	Unable to ambulate	Dyspnoeic, requires assistance	Steady gait, no dizziness or pre-op level
Nausea & vomiting	Continues after repeated treatment	Moderate, treated with IM medications	Minimal, treated with PO medications
Pain (Acceptable to the patient; controlled with PO meds)	-	No	Yes
Surgical bleeding	Minimal, no dressing change required.	Moderate, up to two dressing changes.	Severe, more than three dressing changes.

Score of 9 or more is required to meet the fitness for discharge.

Blood loss estimation

For the assessment of blood loss during the surgery, the blood suctioned from the surgical area was collected in a suction bottle to which heparin was added. Additionally, the nasal gauze packs soaked with blood were also counted. Each gauze strip measured 4 inches long and ½ inch wide holding 2 ml of blood. Partially soaked gauze strip holds 1 ml of blood.

Fromme Boezzart scale

0 = No bleeding.

1 = Slight bleeding: no suction of blood required

2 = Slight bleeding: occasional suctioning required. Surgical field not threatened.

3 = Slight bleeding: frequent suctioning required. Bleeding threatens surgical field a few seconds after suction is removed.

4 = Moderate bleeding: frequent suctioning required. Bleeding threatens surgical field directly after suction is removed.

5 = Severe bleeding: constant suctioning required. Bleeding appears faster than can be removed by suction. Surgical field severely threatened.

Data management and analysis

The variables were entered into SPSS, version 11, statistical software for analysis. The descriptive statistics of the variables studied are represented as two-way tables. The categorical factors are represented by the number and frequency of cases. The continuous variables are represented by measures of central frequency (like mean, median and mode) and deviation (standard deviation and range). The differences in the proportions are tested for statistical significance using non parametric chi- square test for variants measured on nominal scale. When testing for two groups, student “t” test is used to test for statistical significance in the differences of the two means. Line graphs were used to illustrate the hemodynamic monitoring at different time points. Box plot graphs were employed to depict the distribution of other factors in the two groups.

CHAPTER VIII

OBSERVATION AND RESULTS

DEMOGRAPHIC DATA

TABLE I

Age distribution[$]

AGE	SEVOFLURANE	PROPOFOL	p-value
No. of cases	25	25	0.91
Mean	38.16	37.88	
S.D.	8.18	9.97	
Range	24-55	20-52	

[$] *Not statistically significant*

The mean age between the comparison groups are almost similar.

The minimum age taken for the study is 20 and maximum is 55.

TABLE II

Sex distribution[$]

	SEVOFLURANE		PROPOFOL		p-value
	No.	%	No.	%	
Male	20	80	19	76	1.00
Female	5	20	6	24	

[$] *Not statistically significant*

A male preponderance is forthcoming in all the study groups. However, the distribution of sex among the groups is not statistically significant.

TABLE III

Weight distribution[$]

Weight	SEVOFLURANE	PROPOFOL	p-value
No. of cases	25	25	0.91
Mean	63.44	62.2	
S.D.	6.49	6.78	
Range	50-75	50-75	

[$] *Not statistically significant*

The mean distribution of cases by weight was observed to be not statistically significant between the two groups.

TABLE IV

Intra-operative requirement of Nitroglycerine[$]

NTG used	SEVOFLURANE		PROPOFOL		p-value
	No.	%	No.	%	
Yes	7	28	6	24	0.74
No	18	72	19	76	

[$] *Not statistically significant*

Both groups require nitroglycerine intra operatively. There is no statistical significance between the two groups in intra-operative requirement of nitroglycerine.

TABLE IV

Intra-operative hemodynamic parameters

Parameter	SEVOFLURANE	PROPOFOL	p-value
Heart rate			
Before induction	78.44±8.40	78.56±9.29	0.96
After induction	83.88±8.53	81.76±6.89	0.34
After intubation	89.08±7.73	92.32±7.75	0.15
Average intra-op	84.56±4.36	73.93±4.97	0.00*
Immediate post-op	84.48±7.16	79.44±7.97	0.02*

Systolic blood pressure			
Before induction	124.80±10.04	124.40±8.99	0.88
After induction	100.92±10.53	107.60±10.10	0.03
After intubation	125.60±12.13	123.92±15.19	0.67
Average intra-op	91.09±4.80	92.81±4.17	0.19
Immediate post-op	124.24±9.93	119.76±9.19	0.10
Diastolic blood pressure			
Before induction	82.28±6.19	82.16±7.77	0.95
After induction	68.40±8.88	70.08±8.66	0.50
After intubation	85.52±9.66	83.92±12.09	0.61
Average intra-op	60.03±2.87	62.00±2.49	0.01*
Immediate post-op	83.60±6.79	79.04±6.05	0.02
Mean arterial blood pressure			
Before induction	96.44±7.04	96.20±7.79	0.91
After induction	79.24±8.63	82.56±8.64	0.18
After intubation	98.88±10.24	97.36±12.90	0.65
Average intra-op	70.36±3.13	72.26±2.40	0.02*
Immediate post-op	97.16±7.30	92.64±6.47	0.03

* *Statistically significant*

There is a statistically significant difference in the average intra operative heart rate. There is a significant reduction in heart rate in the propofol group when compared to the sevoflurane group. There is a statistically significant difference in the average intra operative systolic blood pressure between the two groups with the

sevoflurane group having a lower value. There is a statistically significant difference in mean arterial pressure. Between the two groups with the sevoflurane group having a lower value.

TABLE VI

Intra operative adverse events[$]

Intra operative problems		SEVOFLURANE		PROPOFOL		p-value
		No.	%	No.	%	
Arrhythmia	Yes	0	0	0	0	
	No	20	100	20	100	_
Hypotension	Yes	6	24	2	8	
	No	19	76	23	92	0.25
Hypertension	Yes	0	0	0	0	
	No	20	100	20	100	_
Tachycardia	Yes	0	0	0	0	
	No	20	100	20	100	_
Bradycardia	Yes	0	0	0	0	
	No	20	100	20	100	_
Ischemia	Yes	0	0	0	0	
	No	20	100	20	100	_

[$] *Not statistically significant*

Intra operative problems such as tachycardia, bradycardia, arrhythmias, ischemia and hypertension are not seen in any of the groups. Hypotension is the commonest recorded adverse event observed in both the groups with six cases in the sevoflurane group and four cases in the propofol group.

TABLE VII

Effect of study drugs on duration of surgery[$]

Duration of surgery	SEVOFLURANE	PROPOFOL	p-value
No. of cases	25	25	0.16
Mean	64.8	71.4	
S.D.	16.99	15.24	
Range	30-90	45-100	

[$] *Not statistically signicant*

There is no statistically significant difference in the duration of surgery between the two groups.

TABLE VIII

Evaluation of surgical field by surgeon

Fromme Boezzart scale	SEVOFLURANE	PROPOFOL	p-value
No. of cases	25	25	0.00*
Mean	1.96	1.16	
S.D.	0.2	0.37	
Range	1-2	1-2	

***** *Statistically significant*

Propofol group provided a better Fromme Boezzart score in the evaluation of surgical field. This is a statistically significant difference.

TABLE IX

Intra operative blood loss

Blood loss	SEVOFLURANE	PROPOFOL	p-value
No. of cases	25	25	0.02*
Mean	156.8	102	
S.D.	91.33	58.83	
Range	25-330	10-210	

* *Statistically significant*

Propofol group shows statistically significant decrease in the intra operative blood loss.

TABLE X

PADSS - 24 hours post op

PADSS – 12 pm 1st POD	SEVOFLURANE		PROPOFOL	
	No.	%	No.	%
9	25	100	25	100

There is no statistically significant difference between the two groups.

All patients are fit to be discharged 24 hours after surgery.

CHAPTER IX

DISCUSSION

The primary goal of an anaesthetist in FESS procedure is to provide better surgical access, a blood less operating field, conduct of balanced anaesthesia and prompt recovery. To provide a blood less operating field, we need to control the three sources of bleeding – venous, capillary and arterial. In our study, we have used methods to control bleeding from all the three sources. Proper positioning of the patients, for example in head and neck surgeries, a 15-20^{o} reversed Trendelenberg position promotes venous drainage and reduces bleeding. To control the capillary bleeding, the surgeon uses nasal packs with decongestants like oxymetazoline and infiltrates the field with 1% lignocaine with 1:1,00,000 adrenaline for vasoconstriction. We have used this concentration for standardization. In order to control the arterial bleeding, we have used induced hypotension to reduce the mean arterial pressure to around 70 mmHg.

This study compares the efficacy of sevoflurane and propofol as maintenance anaesthesia in reducing intra operative blood loss and improving the surgical field.

As per the study, the two groups did not demonstrate any statistical significance in demographic distribution.

Both the groups are comparable in their resting heart rate and blood pressure. All the patients are normotensive. There is no statistically significant difference between the groups.

Since both the groups receive the same mode of induction with similar dosage of drugs, intubation responses also did not vary between the two groups.

Intra operative mean arterial pressure was maintained around 70 mmHg with the use of a deep plane of anaesthesia with either sevoflurane or propofol. In patients who failed to achieve the targeted mean arterial pressure, the vasodilator nitroglycerine was used. In the sevoflurane group, the patient was handed over to the surgeon only after a minimum of 15 minutes to allow equilibration of alveolar and brain partial pressure to the concentration set in the dial. The flaw in our study is the non usage of end tidal agent analyzer due to non availability in our setup. Intra operative mean arterial pressure was found to be lower in the sevoflurane group. **Shigeiki et al**[11], in their study found that the mean arterial pressure was

lower in the propofol group than in the sevoflurane group. The findings in our study did not concur with this. The current study also showed that the diastolic blood pressure was lower in the sevoflurane group.

Nevertheless, the intra operative heart rate was found to be lower in the propofol group. This finding concurred with the results of the study by **Steinmetz et al[12],** which found that the propofol infusion group was associated with lower heart rate than sevoflurane group. This finding also concurs with the results of the study by **Zeliha et al[13],** in which preoperative heart rates are lower in the propofol infusion for maintenance of anaesthesia group.

A comparable number of patients in both the groups required nitroglycerine infusion for maintaining the intra operative mean arterial pressure at around 70 mmHg. There was no statistically significant relevance in these findings.

Intra operative problems like hypertension, arrhythmia, tachycardia and ischemia were not encountered in either of the groups.

Hypotension was the most common intra operative problem encountered with 6 (24%) patients in the sevoflurane group and 2 (8%) in the propofol group. Our findings did not concur with the results of the study by **Jellish et al**[14], which states that both propofol and sevoflurane groups are hemodynamically stable throughout the study period. The results of the study by **Watson et al**[15], that cardiovascular stability was good and comparable in both the groups seems to support our study.

There is no statistically significant difference in the duration of surgery between the two groups.

In the evaluation of the surgical field by surgeon using Fromme Boezzart scale, propofol group provided a better score over sevoflurane group. This finding concurs with the results of the study by **Eberhart et al**[7].

There is a marked difference in the intra operative blood loss between the two groups with propofol group providing the least blood loss. The lower intra operative heart rate leading to lower cardiac output in the propofol group may be accounted for the lower blood loss and better surgical field.

All the patients in both the groups met the discharge criteria 24 hours after the surgery. Discharge criteria was assessed only after 24 hours because in this institute, patients undergoing FESS surgery have their nasal packs changed to evaluate for bleeding. Hence they are not discharged prior to 24 hours.

CHAPTER X

SUMMARY

The prospective randomized study aimed to compare the effects of sevoflurane and propofol as maintenance anaesthetics on peri-operative characteristics of patients undergoing FESS procedure under general anaesthesia and induced hypotensive anaesthetic technique.

Important conclusions from this study include

1. Both drugs provide good to excellent conditions during maintenance of anaesthesia.

2. Sevoflurane lowered intra operative blood pressures better than propofol anaesthesia.

3. Propofol produces lower intra operative heart rate than sevoflurane anaesthesia.

4. Sevoflurane anaesthesia causes more incidence of intra operative hypotension than propofol based anaesthesia.

5. Propofol anaesthesia provided better visualization of the surgical field assessed by the surgeon using Fromme Boezzart scale.

6. Propofol based anaesthesia reduces intra operative blood loss compared to sevoflurane anaesthesia.

7. Propofol and sevoflurane based anaesthesia causes easily reversible induced hypotension. It did not extend into the post operative period nor was there any rebound hypertension.

8. A few patients in both sevoflurane and propofol groups require vasodilator nitroglycerine to achieve the target mean arterial pressure.

9. Lack of usage of end tidal agent analyser was a limiting factor in the study.

10. Balanced anaesthesia technique with meticulous attention to patient's needs is the single most important factor in production of controlled hypotension during surgery with propofol anaesthesia reducing the intra operative blood loss.

CHAPTER XI

CONCLUSION

Sevoflurane and propofol based anaesthesia produce comparable and excellent intra-operative conditions during anaesthesia for FESS.

Sevoflurane group produced superior blood pressure control, while propofol had lower and more stable heart rates.

Propofol based anaesthesia had lesser intra operative blood loss and provided better visualization.

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PROFORMA

DEPARTMENT OF ANAESTHESIOLOGY,

MADRAS MEDICAL COLLEGE, CHENNAI.

A COMPARITIVE EVALUATION OF PROPOFOL AND SEVOFLURANE BASED ANAESTHETIC TECHNIQUE ON PERI OPERATIVE PARAMETERS OF PATIENTS UNDERGOING FESS UNDER HYPOTENSIVE ANAESTHESIA

Name:

Age/Sex:

Diagnosis:

Surgery:

Pre op assessment:

Airway:

Comorbid illness:

Drug therapy:

ASA	Height	Weight

Name
Age/Sex
Diagnosis
Surgery
Pre op assessment
Airway
Comorbid illness
Drug therapy

ASA	Height	Weight

INVESTIGATIONS

Hb	PCV	BT	CT	Sugar	Creatinine	Platelets

PREMED

Alprax 5 mg	Time	Ranitidine 150 mg	Time

Nasal Pack	Drug	Volume

IV Access	18 G

MONITORS

ECG	NIBP	SPO_2	Temp.

GA with CV

Induction			ETT
Fent 2µg/kg	Propofol 2.5mg/kg	Rocuronium 0.6mg/kg	

Infiltration	1% Lignocaine with 1:100000 Adrenaline	Volume

Maintenance

Sevo																												
Fent	**.5µg/kg**																											
Roc	**.2mg/kg**																											
NTG																												

INTRA OP EVENTS

	Episodes	**Treatment**
Hypotension MAP < 60		
Hypertension MAP > 80		
Bradycardia HR < 50		
Tachycardia HR > 150		
Desaturation, Ischemia		

INTRA OP HEMODYNAMIC PARAMETERS

HR																													
SBP																													
DBP																													
MAP																													
SPO_2																													

Duration of surgery	

Total blood loss	**Suction**	**Pads/Gauze**	**Surgical field**

Reversal		**Extubation**
Neostigmine	**Glycopyrrolate**	

Post Op:

HR:

BP:

PADSS :

Consciousness:

CVS:

RS: